A CASE OF MYASTHENIA GRAVIS WITH AUTOPSY.*

By Charles W. Burr, M.D.,

OF PHILADELPHIA.

PROFESSOR OF MENTAL DISEASES IN THE UNIVERSITY OF PENNSYLVANIA.

(From the William Pepper Clinical Laboratory,
Phœbe A. Hearst Foundation.)

This case is of pathologic interest because it adds another to the relatively large, though absolutely small, number in which the thymus gland was either persistent in the adult, or persistent and diseased, and associated with lymphoid infiltration of the muscles. It is interesting clinically because the patient exhibited visual symptoms most frequently met with in, and at one time regarded as pathognomonic of, hysteria; namely, contraction of the fields of vision, partial reversal of the red and blue fields, and later color confusion. Similar disturbances of vision have not been reported in any other case. In not a few, however, the fields were not taken, the writers simply stating that vision was normal. In the first case which I reported the fields were concentrically somewhat diminished, but there was not reversal. Myasthenia gravis is so almost exclusively a motor disease that I am not at all sure that the visual symptoms in this case were more than coincidental. The patient presented no other hysterical symptoms, the course was typical of myasthenia, and the result was death. That reversal of the fields may occur in organic disease and in functional troubles other than hysteria is of course well known.

The history of the case is as follows. The patient was a white unmarried brakeman, thirty years old. His father had died from a gunshot wound and his mother in childbed. He had had malarial fever in 1894 and gonorrhea about ten years ago, but never syphilis. He had always been robust. He first visited Dr. Stahl, through whose courtesy I saw him, and by whose kindness I am permitted to make this report, on October 14, 1900, complaining that for about two months he had

---

* Read at the Philadelphia Neurological Society, Feb. 23, 1904.

had a great deal of headache, that about October the first the right eye began to droop, especially in the evening, and that at the same time the muscles of the jaw grew weak, the weakness soon spreading to the shoulders and arms, so that he had trouble in putting on his coat, shaving and buttoning his collar. At the same time vision became slightly blurred and there was some napeache and quite a little vertigo.

On examination he looked like a muscular, well-nourished man. He walked well with good firm tread, but could not raise the arms above the shoulders and any movement of the arms or hands caused great tire. Sensibility to touch, pain, and temperature was normal. The knee-jerk was present and slightly more marked on the left, but not spastic on either side. There was neither ankle clonus nor Babinski's jerk. The irides reacted promptly to light and with accommodation. He complained of diplopia. The bladder and rectum were under complete control. Speech was slow, the voice weak, and it tired him greatly to talk, but there was no aphasia or vocal palsy. When sitting upright he could swallow well, but if he leaned forward solid food stuck in his throat. He could swallow liquids without difficulty. His mental state was good. The lungs were normal. There was a blurring of the first mitral sound and the second was sharp and somewhat accentuated. The aortic sounds were normal. The area of cardiac dulness was not enlarged. He was sent to Dr. Stahl's wards at St. Agnes Hospital and for a time his muscular weakness varied greatly from day to day and even at different times of the same day, but the general trend was constantly downward. For example, by November he had great trouble in swallowing, and liquids were regurgitated through the nose. By November 20 he could scarcely get out of bed and could walk only a few halting, staggering steps. All movements of the arms were very weak and after a few efforts they would fall helpless by his side. He could hold his head up for a few moments only. In fact all effort so tired him as to cause a total though transient inability to contract the muscles he had just used. I am not sure, but think, that the use of any one group of muscles produced some weakness in others. There was no ataxia. The knee-jerk was very capricious. Sometimes a blow upon the tendon would cause a slight jerk, sometimes not. Several taps might cause no movement and then a final stroke produce a slight response. The knee-jerks were never exaggerated. At rest there was marked ptosis, most marked in the right eye. He could open the eyes widely but could not hold them so. There was but little, and soon no, power of voluntary contraction of the frontal muscles. He could move the tongue in all directions. The face was quite expressionless at rest, smoothed out, masklike. I did not notice the nasal smile mentioned by Gowers. There was no muscular wasting in the face, tongue, or extremities. There was some tremor of the tongue on extension. He could turn the head, lying in bed, much better to the left than to the right. Faradic response was slower than in a healthy person. Sensibility was still preserved and he still controlled the bladder and rectum well. For some days before death he could swallow only a little mushy and liquid food. There was no fever at any time. On November 23, 1900, he began to complain of difficulty in breathing, or rather of a sense of suffocation. This continued till November 27, when, still conscious and mentally clear, he died.

His eyes were examined several times by Drs. F. M. Perkins and P. A. Bly. The fundi were normal. At the first examination on October 29 there was some contraction of the right field with, in places, an extension of the red field beyond the blue. The left field was at this time practically normal. On November 10 both fields were quite a great deal contracted, but the red extended beyond the blue in only one place in the right field. On November 17 and thereafter he confused colors a great deal, calling red blue and sometimes white, and blue white. This was not due to speech defect or mental hebetude, but to inability to appreciate color. There was some weakness of some of the extra-ocular muscles, but the notes of this part of the examination were lost and no positive statement can be made as to the muscles affected.

The urine was normal through the course of the disease.

The necropsy was made the same day the patient died by Dr. Joseph Walsh, the pathologist to St. Agnes Hospital. External examination of the body revealed nothing, and on cutting through the skin there was found the normal amount of fat. The spleen was of normal size and consistency but on section the lympth follicles seemed a little more striking than usual. Microscopic examination showed no disease. The kidneys were a little enlarged and congested, and microscopic sections showed hyperemia and slight cloudy swelling. The adrenal glands were normal. On opening the thorax the thymus gland stood out prominently. It weighed 22 G. It contained an encapsulated chronic abscess. (Dr. Walsh informs me that this was not the creamy fluid found in degenerating thymus glands, but a true abscess.) The pus was cultured on agar and bouillon and smears were made. The smears showed no organisms with the common stain. The cultures showed the bacillus coli communis which was probably a contamination. Dr. D. J. McCarthy and I carefully examined the muscular and nervous systems. There was no change visible to the naked eye in either the brain or cord. For microscopic examination the hemalum-eosin, Marchi, Weigert sheath, Nissl. and the fresh osmic acid methods were employed. The cranial nerves and the spinal cord were entirely normal on microscopic examination. The only changes found in the brain were small hemorrhagic areas in the region of the aqueduct of Sylvius. These were recent, without other evidence of inflammatory reaction, and may therefore be considered to have been either agonal or to have occurred a very short time before death. The muscles, Fig. I, the tongue and a bit of one of the pectorals, showed a distinctly pathological condition. The hemalum-eosin method revealed areas of small, round, nuclear infiltration betwen the fasciculi of muscle tissue, triangular or quadrangular in shape. The cells were of two types. The larger were small, round, deeply staining nuclei, one and one-half times the diameter of a red blood corpuscle, and strikingly resembled the cells of the thymus gland. They were, however, somewhat larger than the cells of a thymus of a fetus which was examined as a control. There were beside these, small round cells having nuclei of about the same size but with quite large cell bodies and staining a bright red with eosin. A few small round cells were seen scattered along the blood vessels in the reticular tissues. The cellular accumulations in the tongue were similar to those in the other muscle, except that the nodes were not so large. The types of cell appeared to

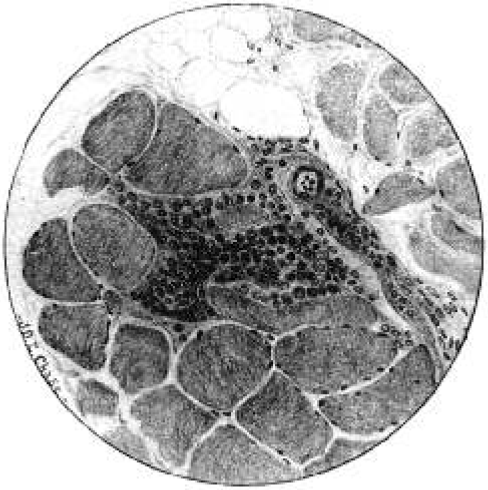

Fig. 1.

be the same. To sum up the lesions found were perivascular accumulations of free red blood cells in the region of the red nucleus and a lymphoid infiltration of the muscles.

In January, 1901, I published with Dr. D. J. McCarthy[1] the report of a case of asthenic bulbar palsy in which the thymus gland was persistent. I failed to appreciate at that time that there could be any relation between the thymus gland and asthenic palsy and regarded the pathologic finding as a mere coincidence without significance. Weigert was the first to point out that there might be a connection between the thymus gland, lymphoid infiltration of muscles, and myasthenia. In 1901[2] he reported a case with sarcoma arising almost surely from the remnant of the thymus and accompanied with an infiltration of the muscles by lymphoid cells. Later Link[3] reported lymphoid infiltration in the muscles with a persistent thymus. Hoedelmoser[4] reported a case with persistent thymus. Recently Henry Hun[5] has published an elaborate account of a case in which there was a lymphosarcoma of the thymus with the same muscular infiltration described by Weigert. He also gives an excellent bibliography. Goldflam[6] found a sarcoma of the lung the origin of which may have been in the thymus gland, and lymphoid infiltration of the muscles. In Oppenheim's[7] case, which seems to have been one of myasthenia gravis, there was a sarcoma of the anterior mediastinum.

The morbid anatomy and pathology of myasthenia gravis remain to be discovered, but till now the most frequent lesions found have been a persistent, or persistent and diseased, thymus and lymphoid infiltration of the muscles. The changes which have been found in the nervous system, slight lesions of the cranial nerves and their nuclei, microscopic hemorrhages in the floor of the fourth ventricle, and congenital abnormalities of the cord are not sufficient to explain the symptoms.

[1] The American Journal of the Medical Sciences, January, 1901.

[2] Neurologisches Centralblatt, Band XX., p. 597, 1901.

[3] Deutsche Zeitschrift für Nervenheilkunde, Band XXIII., p. 114, 1901.

[4] Zeitschrift für Heilkunde, Band XXXIII, p. 279, 1902.

[5] Albany Medical Annals, January, 1904.

[6] Neurologisches Centralblatt, Band XXI., p. 97, 1902.

[7] "Die Myasthenische Paralyse," Berlin, 1901.